In the Specification:

Please amend the specification as shown:

Please delete the paragraph starting on page 21, lines 26-31 and replace it with the following paragraph:

Fig. 1 shows the MPK4 genomic fragment **(SEQ ID NO: 1)** amplified by primers (18mers at ends of this sequence: no name BamH1 linker and p3y). The sequence is the complement of nt4700-7918 of BAC IG002N01 (GENBANK/EMBL accession AF007269, NID 2191126 deposited 12/6/97. According to accession, gene starts at 130 of this sequence. Underlined are exons from cDNA (accession D21840, NID 457399), bold are start, stop, and > is Ds insertion site;

Please delete the paragraph starting on page 25, line 30, bridging page 26 and replace it with the following paragraph:

Total RNA was prepared for RNA gel blot hybridisation using standard protocols (RNA-gents®Total RNA, Promega). Probe templates were amplified by PCR from cDNAs or genomic DNA with primer sequences from: *MPK4* (GI:457399), PR1 (GI:4454853), P-1,3-glucanase or PR2 (GI:166637), PR5 (GI:6646759), *PDF1.2* (GI: 4759674), *THI2.1* (GI:1181530) and elongation factor 1α control (GI:16260). For cDNA microarray analysis, total RNA from 2g of 18 day old, soil grown wild type and *mpk4* was extracted using Trizol Reagent (Life Technologies). Poly(A)$^+$ RNA was purified from 200µg total RNA with 2µg of Dynabeads Oligo(dT)$_{25}$ **(SEQ ID NO: 8)**(Dynal) cDNA microarray production, preparation of fluorescent probes and microarray hybridisation and scanning have been described previously (Ruan et al., 1998). The hybridisation experiment was performed twice using microarrays hybridised to cDNAs from two samples each of *mpk4* and wild type mRNA.

Please delete the paragraph starting on page 29, lines 4-20 and replace it with the following paragraph:

Three approaches demonstrated that this insertion was responsible for the *mpk4* phenotype. First, revertants were generated by *Ds* excision following crosses to a line expressing *Ac* transposase. This identified wild type F3 plants homozygous for kanamycin resistance. Genornic fragments were amplified from revertants, wild type, and *mpk4*. Sequencing revealed that *Ds* had created an 8bp

target site duplication on insertion in the *MPK4* intron, and that a 7bp footprint remained after *Ds* excision to restore the transcription unit (Fig. 2A, SEQ ID NO: 5-~~10~~7). Thus, transposition away from MPK4 is linked to reversion of the dwarf phenotype. Second, RNA blot hybridization showed that *mpk4* homozygotes did not accumulate detectable *MPK4* mRNA, in contrast to wild type (Fig. 2B) as well as the revertant (not shown). Third, *mpk4* mutants were rescued by transformation with a 3.3kbp fragment containing *MPK4* and 1150bp of 5' upstream and 506bp of 3' downstream sequence. In addition, *mpk4* was complemented with the same genomic fragment containing a triple HA-epitope tag at the C-terminus of MPK4. Western blotting and in gel kinase assay showed that MPK4 is active in wild type plants (Fig. 2C). In contrast, equivalent levels of a catalytically inactive, HA-tagged MPK4 containing two mutations in activation loop residues (T201A/Y203F) had no effect on the *mpk4* phenotype (AEF-HA; Fig. 2C). These results demonstrate that the *mpk4* phenotype is caused by loss of MPK4 kinase activity.